These features are relevant to the phenomenon of UH-induced platelet activation because of the following.

1. UH is used in patients with vascular disease. As Sobel et al[1] point out, platelet activation in these patients is undesirable. The clinical and experimental situations described above provide an opportunity to assess the clinical relevance of direct UH-induced platelet activation.
2. The findings described above provide models where the molecular basis of the mechanism described by Sobel et al[1] can be assessed. For example, it is expected that LMWH will bind less to Gp IIb/IIIa than UH. Similarly, UH binding and/or outside-in signaling should be enhanced in patients with peripheral vascular disease, especially if they demonstrate platelet hyperactivity.

Direct platelet activation by UH may reduce the benefit accrued from the use of this anticoagulant. This concept is likely to be further clarified now that Sobel et al[1] have worked out the process of direct UH-induced platelet activation.

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Regarding "The 50th anniversary of abdominal aortic reconstruction"

In his historical review of abdominal aortic reconstruction (J Vasc Surg 2001;33:895-8), Dr Friedman has written a tribute of Dubost and Oudot with empathy, but, to quote Goethe, "Often when primacy is claimed, the pleasure of discovery is spoiled because we are not the first." Their excellent work[1] has been well recognized, while that of Freeman,[2] who preceded them, has been ignored, except by Bergan.[3]

Many surgeons, on returning home at the end of World War II, found conditions ideal for laboratory and clinical research. The scene was thus set for the explosive development of vascular surgery during its golden decade (1946-1956). In San Francisco, at the medical school of the University of California, a small group (Gilfillan, Wylie, and Leeds) commenced, under the guidance of Dr Freeman, to work on various problems in vascular surgery. These were wonderful years, filled with the joy of learning, but we soon found out that there is no easy road to first discoveries. As F. Paiz expresses it, "Traveler, there is no path, the path is made by walking."

Dr Freeman asked me to study (1) the possibility of developing an experimental model of aneurysm and (2) the bursting pressures of arteries and veins. At that time, in our operative approach to abdominal aortic aneurysms, we had tried ligation, wiring, and cellophane wrapping, but the results and complications were discouraging. As the data on bursting pressures in the experimental animal suggested that the iliac vein would withstand arterial pressures, on February 12, 1951, we resected an aneurysm and replaced it with a vein graft taken from the patient's left common iliac vein and its bifurcation. Six hours postoperation the patient died. We were unable to obtain an autopsy, but the sudden death, we felt, was due to rupture of the vein graft. Therefore, on February 26, 1951, when we were presented with a 55-year-old man with a large symptomatic aneurysm, Freeman modified the operation by opening the aneurysm longitudinally, and a vein graft, consisting of the patient's left common iliac vein and its bifurcation, was sutured within the aneurysmal sack to the aorta and iliac arteries. The aneurysmal sack was then closed around the graft.

The 15-month follow-up, including an aortogram at 4 months, showed an excellent result. This case was presented at the first meeting of the International Society of Angiology (now the American Association for Vascular Surgery) on June 9, 1951, and published in *Angiology* in December 1951.[4]

The obvious problem with this technique was the extra time and trauma needed to obtain the autologous graft. A cadaver graft seemed the solution, but Freeman, on the basis of earlier studies,[5] refused to consider it, as he felt a homologous graft would not hold up over time.

Then in June of 1952, Freeman returned from a trip to the East Coast with the exciting news of Voorhees' dramatic breakthrough[6] and a piece of Vinyon-N cloth, given to him by Blakemore, and aortic aneurysm surgery was really on its way, for now we had a readily available graft for the inlay technique, which was used, from then on, by Freeman and myself and was eventually popularized by Creech.[7] Our inlay technique was a simpler one than the excisional one, later developed by Dubost, which required "a sometimes difficult and hazardous dissection."

This explosive development in vascular surgery was not localized, but generalized throughout Europe and America. Should

we not then say that all who were part of that generation were part of the First?

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Competition of interest: FHL was associated with Dr Norman E. Freeman in research, teaching, and private practice of vascular surgery from July 1946 to September 1957.

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Regarding "Preprocedural risk stratification: Identifying an appropriate population for carotid stenting"

We read with interest the article by Dr Ouriel and his colleagues from The Cleveland Clinic Foundation (J Vasc Surg 2001;33:728-32). There is a recent nationwide explosion of interest in treating patients at high risk for carotid endarterectomy (CEA) with percutaneous angioplasty and stenting (CAS). Similar to the conclusion arrived at by Ouriel et al, we randomly assigned patients with significant baseline comorbid conditions (cardiac, recurrent stenosis, radiation stenosis, and contralateral occlusion) to CEA or CAS.

Significant numbers in both treatment groups had grade 3 (SVS/AAVS) cardiac (CAS 72%, CEA 60%; $P = .44$) and hypertensive (CAS 82%, CEA 80%; $P = .65$) risk factors. Contralateral carotid disease (>50% stenosis, occlusion, or prior endarterectomy) was present in 64% of the CAS group and in 60% of the CEA group. Significantly more reversible cardiac events (hypotension/bradycardia requiring ≥24 hours of pharmacological support) were observed in the CAS group (CAS 73%, CEA 20%; $P = .03$). Major adverse in-hospital events were noted in one patient in each group (CAS, myocardial infarction; CEA, death). Local complications were observed in one from each treatment group (CAS groin hematoma; CEA, recurrent laryngeal nerve paralysis). The duration of postoperative stay did not reach statistical significance (CAS, 2.1 ± 1.4 days; CEA, 1.8 ± 1.1 days). However, four patients in the CAS treatment group were readmitted within 1 month (congestive heart failure, 1; myocardial infarction, 1; rest pain, 1), compared with no new events in the CEA group.

A disturbing number of patients in the CAS treatment group experienced profound and prolonged hypotension and bradycardia induced by forced dilatation of the carotid sinus. These physiological changes were not well tolerated by patients with underlying cardiac risk factors. Based on the findings of our study population, a treatment algorithm is currently used before randomization (Fig 1). Routine cardiac stress evaluation and coronary revascularization if possible significantly reduced the adverse cardiac events noted following CAS. Patients with uncorrectable cardiac disease are currently not offered CAS at our institution.

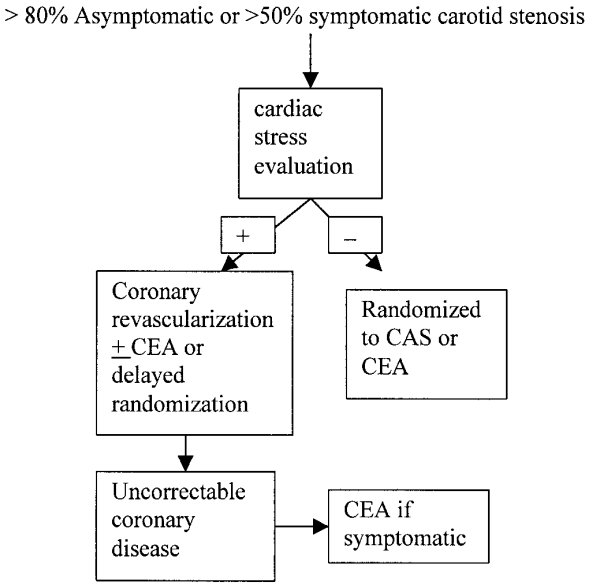

Fig 1. Treatment algorithm used before randomization.

Fig 2. Isolated stenting of the ICA.

Our findings are similar to a study by Gil-Peralta et al[1] who also reported a high incidence of cardiovascular effects: hypotension (54.1%), bradycardia (67.1%), and asystole in 25.9% of treated patients. Accurate stent sizing using an intravascular ultrasound to measure the normal caliber of the distal internal carotid artery (ICA), combined with isolated ICA stent placement (Fig 2) has also decreased the incidence of post-CAS bradycardia and hypotension. Evolving technology, such as the development